THE TREATMENT OF SPASTICITY AND ATHETOSIS BY RESECTION OF THE POSTERIOR SPINAL ROOTS.[1]

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Cases of spasticity are very numerous, and in some instances the spasticity is far greater than the motor palsy; indeed, if the implication of the pyramidal tract be slight, the spasticity, in the opinion of some, is greater than when the degeneration is intense. Spasticity is affected by various conditions. Sensory irritation, such as cold, faradic electricity, handling of the body, or voluntary movement increases the spasticity; and sleep may diminish it; so that spasticity may be less in the morning than in the evening. These effects are produced through the peripheral nerves.

Irritation of peripheral sensory fibers, as in joint disease, may increase the tonus and cause contracture. It is common to find the tendon reflexes exaggerated in the wasted limb in arthritic muscular atrophy.

Athetosis is essentially a form of spasticity, differing from ordinary spasticity chiefly in the varying degree of tonicity in the different muscles. An athetoid limb is one necessarily spastic, and first one group of muscles, then another, exhibits a predominance of the spasticity. This view has been shared recently by Schwab and Allison;[2] they state that athetosis and spastic conditions of organic origin are essentially similar processes; that athetoid movements may be regarded as variants of permanent tonic spasticities.

In order that we may have clearly in mind the rationale of the method of treating spasticity by resection of the posterior roots, it may be well to give at this juncture an elementary sketch of the chief factors concerned in spasticity.

Muscular tonus depends on the integrity of the peripheral sensory nerve fibers, of the posterior roots, of the cells of the anterior horns, and of the peripheral motor nerve fibers. Fibers pass through the brain and cord and exert a controlling influence over the spinal reflexes. These fibers have an inhibitory effect, and act as a damper on the spinal reflexes. In contrast with these are possibly other fibers whose function is to accelerate the tonus, "bahnende" fibers of the German writers. It may be partly from irritation of the latter that in slight lesions of the pyramidal tracts tonicity may be greater than in complete destruction of these tracts.

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[1] A lecture delivered at the University of Pennsylvania during "Home Coming" week, March 29, 1910.

[2] Journal of Nervous and Mental Disease, August, 1909; Journal of the American Medical Association, February 12, 1910, p. 551.

The inhibitory effect of the cerebrum on the spinal reflexes is probably accomplished through the pyramidal tract. Afferent impulses are passing constantly from all parts of the body below the head to the spinal cord, and exert an influence on the cells of the anterior horns of the spinal cord, by means of which motor impulses are sent out to the muscles. It is, indeed, fortunate for us that many of these reflex acts do not enter our consciousness. When standing in a rapidly moving car we may be engaged in reading, and yet are constantly and unconsciously altering the tonicity of opposing groups of muscles. A wooden figure of the same proportions as the human body placed in a similar position would soon fall over. During life our muscles are constantly in a state of tonicity suitable for the various functions they are called upon to discharge, but in disease this condition of well-adjusted tonicity may be altered. A soldier standing "at attention," as Strümpell has said, exhibits the most perfect movement.

When some lesion damages the pyramidal tract the inhibition of the brain is removed and the spinal reflexes are left without control. The desideratum would be the restoration of the pyramidal tract, but that manifestly is impossible. The next best thing is to remove some of the afferent peripheral impulses, so that the cells of the anterior horns may not be kept in a state of overexcitement. It is possible that in lesions of the pyramidal tract the excessive tonicity is caused by the cerebellum, and theoretically it might be desirable to operate so as to influence the action of the cerebellar nuclei. An operation may be devised some day with this end in view, although at present it does not seem possible, and yet in 1905 the proposal of division of the posterior roots to influence spasticity met no response.

In this connection I quote from J. S. Risien Russell's Lettsomian Lectures:[3] "As sensation does not accrue as a result of all peripheral stimuli which evoke a central (reflex) action, Sherrington adopts 'receptor' as the neutral term to apply to the peripheral apparatus, which receives the stimulation. He points out that there are two main fields of distribution of the receptor organs—a *surface field,* which is situated at the surface layer of the organism, but external and internal, which he speaks of as exteroceptive and interoceptive respectively; and a *deep field,* which is situated in the tissues of the organism beneath the surface layer, which he designates proprioceptive. The stimulations which occur in the deep field are traceable to actions of the organism itself, as opposed to stimuli received from without. Hence the term 'proprioceptor,' which he has applied to these stimuli, which are brought about largely, as he says, through the agency of mass, with its mechanical consequences of weight and inertia, and also largely through mechanical strains and alterations of pressure, resulting from contractions and relaxations of muscles. Among the functions subserved by the receptors of the proprioceptive, or deep field, is that of maintaining a state of tonic reaction in the skeletal muscles, which Sherrington regards as concerned with the maintenance of attitude. . . .

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[3] British Medical Journal, February 19, 1910, p. 425.

"Bouché has determined that fits induced by intravenous injection of absinthe in an animal deprived of one cerebral hemisphere were clonic and mainly flexor in type from the intact cerebral hemisphere, whereas the fit on the other side was tonic and chiefly extensor. Section of the mesencephalon during the fits abolished the clonic discharges, and only allowed the tonic to continue. Even more striking are the results of Leonard Hill, who was able to alter the character of the fit from tonic to clonic at will, according as he interrupted the arterial supply to the cerebrum by clamping the vessels or allowed the blood charged with absinthe to reach the brain. . . .

"Sherrington has shown that removal of the cerebrum results in what he speaks of as decerebrate rigidity, which fully accords with Dr. Jackson's view of cerebellar influx. Moreover, he found that this rigidity could be abolished, and was replaced by flaccidity in the homolateral limbs on hemisection of the spinal cord in the cervical region, which makes it evident that tonus is derived from the mesencephalon or cerebellum. Sir Victor Horsley, acting on Dr. Jackson's suggestion, divided the mesencephalon at the level of the intercollicular groove, and thus allowed of decerebrate rigidity appearing. Removal of successive sections of the cerebellar cortex produced no appreciable effect on the hypertonus until the sections involved the intrinsic and paracerebellar nuclei [nuclei of Deiters and von Bechterew]. A large horizontal lesion, separating the dorsal half of the cerebellar cortex without involving the nuclei, was also made three weeks before the division of the mesencephalon, again without any appreciable effect on the decerebrate rigidity which resulted in these animals, as compared with that which obtained when a similar section of the midbrain was performed in normal animals. Thiele, in his researches undertaken with a view to determine the source from which the hypertonus which constitutes Sherrington's decerebrate rigidity is derived, found that the chief source of this energy was at the level of the paracerebellar nuclei. Successive lesions of the mesencephalon, commencing in front and proceeding caudalward, failed to affect the decerebrate rigidity until the nuclei were reached. The experimental evidence is thus convincing, both as proving that the nuclear region of the cerebellar complex is the source of the energy, which constitutes the cerebellar influx, and as thus confirming the view enunciated by Dr. Jackson more than thirty years ago. . . .

"All the facts that have been established in regard to the cerebellum are in accord with a view that its cortex is concerned with the reception of afferent impressions which are derived from deeper structures like the muscles and joints, as well as those which the organ receives from the labyrinth, and that efferent impulses are thus evoked from its muscles, which are responsible for that degree of tonus in the general musculature of the organism which is essential for the maintenance of attitude, and which makes it possible for coördinated movements to be executed."

These important statements show that tonicity of muscles depends on the cerebellum, and probably on its nuclei, and less so on the cerebrum. The connections of peripheral afferent fibers with the cerebellum is by means of the posterior roots. It is probable that in excessive tonicity destruction of the cerebellar nuclei would produce flaccidity. By cutting the posterior roots from a spastic limb we weaken or destroy this spasticity by removing the impulses to the cerebellum, on which organ it depends, and therefore the operation has a logical basis. Were we in a position to increase the cerebral inhibition over the motor tracts, we should not resort to resection of the posterior roots.

Various attempts have been made to overcome spasticity. In a paper read in 1905 with Dr. Frazier[4] before the New York Neurological Society on the treatment of cerebral palsies and athetosis, nerve anastomosis was discussed. At that time I recommended the resection of the posterior roots for the relief of spasticity and athetosis, but the idea was so novel it called forth no discussion, and, receiving no encouragement, I hesitated to advise further an operation that seemed too much like an experiment on man, and one not without danger. We therefore contented ourselves with an attempt to relieve spasticity and athetosis by anastomosis of one peripheral nerve with another, hoping that we might satisfactorily diminish afferent impulses, although we knew that we must diminish efferent impulses, but felt that sufficient efferent motor impulses would be left for fair discharge of function. The results seemed at first very promising. The most extreme spasticity and athetosis yielded to this treatment, but the young man on whom the operation was performed deserted the hospital and withdrew himself from our observation. When we saw him again, some three years later, athetosis in the operated limbs had almost disappeared, but contracture had become pronounced and interfered greatly with voluntary motion.

An attempt similar to ours has been made by Nutt[5] recently. This author believes that much may be gained by cerebral rest. His method differs from ours only in uniting the cut ends of the same nerve instead of uniting the cut ends of different nerves.

Schwab and Allison have proposed another method of treatment for the relief of spasticity. Their method is described as follows: "We have been led to devise a method which we shall refer to as muscle group isolation. This implies the isolation of the muscle or the group of muscles which are at fault in the production of contracture, deformity, or athetosis. It is made effective by cutting off from the central nervous system the connection along which the abnormal impulses, active in causing spasticity or athetosis, are transmitted. This is done by a direct attack on the nerve itself, by isolating it, and injecting it with an alcoholic solution. There has resulted in the cases to be described an immediate paralysis of the physiologically stronger group of muscles without interfering with the free muscular use of the antagonists. At this point physiological exercises planned to further strengthen the antagonist may be used."

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[4] Journal of Nervous and Mental Disease, May, 1905; American Journal of the Medical Sciences, March 1906; University of Pennsylvania Medical Bulletin, January, 1910, p 314.

[5] American Journal of Orthopedic Surgery, November, 1909, p. 151.

Schwab and Allison have recently (February 12, 1910) stated that they regard their method as holding considerable promise of relief. They have not attempted to cure spasticity by alcoholic injections into the peripheral nerves. They believe that by their method the peripheral pathway of the spastic impulses being temporarily blocked, their return in the original sum might be lessened.

More promising than any other method for the relief of spasticity has seemed to me for some five years the resection of posterior roots innervating the spastic limbs. He who studies disease probably has many indications justifying this operation. So far back as 1893 or 1894 I observed return of the patellar reflex in tabes after hemiplegia had occurred. This return of reflex irritability indicated that the degeneration of the posterior lumbar roots in this case of tabes had been sufficient to abolish the reflex, but that enough fibers were retained in the posterior roots to permit a return of this reflex when cerebral inhibition over the spinal reflexes was removed by degeneration of the pyramidal tract. The opposite conditions to those described in this case have been observed repeatedly. When degeneration of the pyramidal tracts has been followed by degeneration of the posterior columns extending well into the lumbar region, in posterolateral sclerosis, spasticity and exaggeration of tendon reflexes have given place to flaccidity and diminution of tendon reflexes.

Unquestionably the first operation for the cure of spasticity by division of the posterior roots was made by O. Foerster's directions.[6] His first paper was published in 1908. He relied on Sherrington's experiments on monkeys, by which it was shown that resection of one or two posterior roots produced no anesthesia, and that the sensation of any cutaneous area depends on three roots, and that not until three adjoining roots are cut does anesthesia occur. As yet satisfactory investigations concerning man are wanting, although it has been demonstrated that division of a single posterior root causes no sensory disturbance. Bruns believes that more than three roots may supply any cutaneous area. Foerster has never had more than two adjoining roots divided.

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[6] Zeitschrift für Orthopädische Chirurgie, 1908, xxii, 1 to 3; Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1909, xx, 493.

For the report that he makes in his second paper he selected two cases of congenital spastic diplegia, one case of tuberculous cervical spondylitis, one case of multiple sclerosis, and one case of hemiplegia. After the operation a certain resistance of the muscles persisted for several weeks in passive movement and extension of the limbs. This resistance he attributes to the pain from the irritation of the posterior roots produced by the cutting. He found at first after the operation that the leg was flexed at the knee and the thigh flexed on the hip, rotated inward, and adducted. This position alters as the cut roots degenerate, as does also the resistance of the muscles in passive movement. In spastic paralysis cutting of tendons or muscles seldom is successful. In some cases tenotomy may be necessary after division of the roots. Foerster recommends that the limbs should be placed in some corrective apparatus after division of the roots, to avoid faulty position.

The Achilles reflex was lost after the operation in Foerster's cases. He attributes this to the cutting of the second sacral and possibly also of the fifth lumbar root. It is not known positively in which posterior roots the Achilles reflex arc lies, but Foerster places it from his cases in the first and second sacral and possibly fifth lumbar roots.

Tietze[7] says the operation is not very dangerous. He had five cases with one death, and death in this case was by infection from decubitus. Gottstein had two operations and Küttner five operations and no deaths. It is, however, recognized as a major operation. Tietze refers to another fatal case in which collapse occurred before the roots were cut.

Successful cases treated by this means have been reported by Clarke and Taylor,[8] and Taylor has diminished the seriousness of the operation by devising unilateral laminectomy.

Dr. Frazier and I have reported one case in detail in which the results of the surgical treatment have been most satisfactory. Since the publication of that report the improvement in the patient's gait has been marked. He has been receiving systematic training in walking from Dr. Wm. Burdick.

The patient referred to at a previous part of this paper, on whom nerve anastomosis was performed, has also been treated by this method of resection of the posterior roots by Dr. Frazier, and in this case the cervical roots were cut. Improvement in this case also is distinct, but tenotomy may be needed on account of the shortening of the flexor muscles of the elbows.

Resection of posterior roots is useless when paralysis is intense.

In regard to some of the objections made to the treatment, I acknowledge that the operation is serious; had it not been so I should not have remained contented in 1905 in merely proposing this operation, but because of its seriousness we hesitated to carry out the suggestion. Cortical rest produced by peripheral operations seems to me a condition of little weight in the treatment of spasticity.

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[7] Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1909, xx, 559.

[8] New York Medical Journal, January 29, 1909, p. 215.

The overlap of sensory distributions is considerable, but this does not mean that all the posterior roots of a limb must be cut to lessen spasticity. In the lumbar region the roots at their origin are near together; and in the cervical region, though not so near as in the lumbar region, they are still close to one another, and therefore exposure of a very large area is not necessary. No one would desire complete sensory paralysis from cutting the posterior roots; a paralysis of this kind would be unnecessary, and would probably cause incoördination and defeat the object of the operation. Indeed, because of the overlapping of root distributions loss of sensation is not likely to occur. Considerable impairment of sensation developed in our first case, but this was probably because the man had had previously great impairment of sensation from his injury, and while sensation had returned, the fibers within the cord were far from normal, and therefore after the resection of certain posterior roots too few afferent fibers were left for the maintenance of sensation. Little or no disturbance of sensation was caused in our second case by extensive resection of posterior cervical roots.

In conclusion, I would say that a means has been devised full of promise for selected cases of spasticity and athetosis, and that these conditions which formerly perplexed and baffled us have in a considerable measure yielded to treatment. Undue enthusiasm is to be deprecated, as bringing the method into disrepute. Cases must be carefully selected, and only those are available in which spasticity is great and weakness is comparatively slight. The association of athetosis need not be a deterring factor, as the strength of a limb often is increased by the excessive muscular action of athetosis. As in every new method, caution is needed, and proper valuation can be placed upon the method only when more experience has been obtained.

THE PATHOGENESIS OF THE TOXEMIA OF PREGNANCY.[1]

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It is a curious maladjustment of Nature that places pregnancy among conditions difficult to separate from the pathological. We are accustomed to refer the high morbidity of gestation to the unhygienic influences of modern life, and love to refer to the Indian women who overtook the moving caravans of their tribes after a brief delay for parturition, but history says little about the women who never caught up with the caravan. Today obstetrics in India, China, and Africa deals with the same ponderous process and meets the same formidable hazards as in Europe and America. The comparative study of gestation reveals throughout the animal kingdom a certain lethal tendency reaching its acme in some orders of insects in which ovulation necessarily entails death. Indeed the human family is comparatively fortunate in many of its natural relations to the propagation of the species, while increasing medical intelligence should eventually shield maternity with effective safeguards not provided for the lower animals.

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[1] Read at a joint meeting of the Pathological Society of Philadelphia and the Philadelphia Obstetrical Society, February 10, 1910.

Of the many clinical or more obvious signs of the intrinsic pathology of gestation, some are exhibited by the great majority of gravida, while the notable minority who seem to escape are all liable to the larger accidents of parturition and the puerperal state. Anatomical studies long ago pointed to the kidney as the organ most liable to suffer damage and most responsible for the characteristic grave disorders of gestation. Leyden's "kidney of pregnancy" is firmly established as a very common and in some degree probably constant pathological condition. It consists essentially of a definite, often pronounced grade of fatty degeneration, which forms a background for the more severe lesions which are often encountered in disease, and which include acute degeneration, exudative nephritis, glomerulonephritis, or widespread necrosis, reaching at times almost complete destruction of the organ. In some respects these renal lesions and the associated symptoms are peculiar to pregnancy, and at least in their extent and severity they are not paralleled in any other autotoxic state.

For more than fifty years attention has been directed toward the liver as the probable seat of disease in several of the grave and fatal disorders of pregnancy. I find these indications very clearly apparent in von Frerich's famous work on *Diseases of the Liver.*[2] Here are presented the clinical symptoms and anatomical lesions of acute malignant jaundice, acute yellow atrophy, and eclampsia, with such accuracy and force that the hepatic lesions have never since lost the prominence which his observations helped to give them. It seems to have been the opinion of von Frerichs and Klebs[3] and others that all these diseases with pronounced hepatic lesions were closely related conditions, but in 1896 Jurgens[4] pointed out the specific anatomical changes of the hemorrhagic hepatitis of eclampsia, and he with Lubarsch,[5] Schmorl,[6] and many later writers, have endeavored

[2] Diseases of the Liver, Sydenham Soc. Trans., 1860, 192.

[3] Ziegler's Beiträge, 1888, iii, 1.

[4] Fettemboli u. Metastase von Leberzellen bei Eclampsie; Berl. klin. Woch., 1886, 519.

[5] Die Puerperaleclampsie, Erg. d. allg. Path. etc., 1896, I Abt.

[6] Path.-Anatom. Untersuch. über Puerperaleclampsie, Leipzig, 1893, Arch. f. Gyn., 1902, lxv, 504.